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09/774,809	01/31/2001	Robert McKay	ISPH-0526	2494

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EXAMINER

GIBBS, TERRA C

ART UNIT

PAPER NUMBER

1635

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13

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No. 09/774,809	Applicant(s) MCKAY ET AL.	
	Examiner Terra C. Gibbs	Art Unit 1635	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☒ Responsive to communication(s) filed on 06 March 2003.
- 2a) ☐ This action is **FINAL**. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) ☒ Claim(s) 14,21,22 and 28-34 is/are pending in the application.
- 4a) Of the above claim(s) _____ is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☐ Claim(s) _____ is/are rejected.
- 7) ☐ Claim(s) _____ is/are objected to.
- 8) ☒ Claim(s) 14,21,22 and 28-34 are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on _____ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
- 11) ☐ The proposed drawing correction filed on _____ is: a) ☐ approved b) ☐ disapproved by the Examiner.
If approved, corrected drawings are required in reply to this Office action.
- 12) ☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. §§ 119 and 120

- 13) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
a) ☐ All b) ☐ Some * c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
2. ☐ Certified copies of the priority documents have been received in Application No. _____.
3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).
* See the attached detailed Office action for a list of the certified copies not received.
- 14) ☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).
a) ☐ The translation of the foreign language provisional application has been received.
- 15) ☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

Attachment(s)

- | | |
|--|---|
| 1) <input checked="" type="checkbox"/> Notice of References Cited (PTO-892) | 4) <input type="checkbox"/> Interview Summary (PTO-413) Paper No(s). _____ |
| 2) <input type="checkbox"/> Notice of Draftsperson's Patent Drawing Review (PTO-948) | 5) <input type="checkbox"/> Notice of Informal Patent Application (PTO-152) |
| 3) <input checked="" type="checkbox"/> Information Disclosure Statement(s) (PTO-1449) Paper No(s) <u>2</u> | 6) <input type="checkbox"/> Other: _____ |

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DETAILED ACTION

This Office Action is a response to the Amendment filed January 6, 2003 in Paper No. 10 and the Election filed March 6, 2003 in Paper No. 12.

Claims 1-13, 15-20, 23-27, and 35-40 have been canceled. Claims 14, 21, 22, and 28 have been amended.

Claims 14, 21, 22, and 28-34 are pending in the instant application.

The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

Election/Restrictions

Applicant's arguments regarding the Restriction Requirement filed February 6, 2003 in Paper No. 11 is moot in view of Applicant's amendment to cancel claims 35-40, filed March 6, 2003 in Paper No. 12.

Response to Amendment

The objection to the Specification for references to the American Type Culture Collection is withdrawn in view of Applicant's Amendment to the Specification, filed January 6, 2003 in Paper No. 10.

The obviousness-type double patenting rejection over claims 14, 21, and 22 as being unpatentable over claims 1-23 of U. S. Patent No. 6,221,850 and claims 1-3 of U.S. Patent No.

6,133,246 is withdrawn in view of Applicant's Terminal Disclaimer to obviate a Double Patenting Rejection over a prior patent, filed January 6, 2003 in Paper No. 10.

The obviousness-type double patenting rejection over claims 14, 21, and 22 as being unpatentable over claims 1-2 of U.S. Patent No. 5,877,309 is moot in view of Applicant's Amendment and cancellation of claims 1 and 9, filed January 6, 2003 in Paper No. 10.

The 112, first paragraph rejection over claims 14, 21, 22, and 28-33 is withdrawn in view of Applicant's arguments filed January 6, 2003 in Paper No. 10.

The 35 U.S.C. 102(b) rejection over claims 14, 21, 22, and 28-33 as being anticipated by Semiya et al. (Journal of Biological Chemistry, 1997 Vol. 272:4631-4636) is withdrawn in view of Applicant's Declaration under rule 1.131 filed January 6, 2003 in Paper No. 10.

The 35 U.S.C. 102(b) rejection over claims 14, 21, 22, and 28-33 as being anticipated by Karin et al. [U.S. Patent No. 5837244] is maintained for the reasons of record set forth in the Office Action mailed August 5, 2002 in Paper No. 8.

Applicants argue that Karin et al. discloses protein kinases, JNK1 and JNK2. Applicants further argue that JNK2 is characterized by having a molecular weight of 55 kD and activity similar to JNK1. Applicants also further that Karin et al. teach polynucleotides which encode the JNK polypeptide and a synthetic peptide which binds to the c-Jun N-terminal kinase, JNK. Applicants also argue that Karin et al. do not teach any specific antisense oligonucleotide sequences that are complementary to the sequence of human JNK2 specifically claimed by the

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instant specification. Applicants also argue that Karin et al. provide a general teaching of the possibility of developing antisense agents against JNK2 without providing a reasonable expectation of success or suggestion which render obvious antisense oligonucleotides targeted to human JNK2 protein to treat conditions or disease in animals.

Applicant's arguments have been fully considered, but are not found persuasive because as argued in the previous Office Action mailed August 5, 2002 in Paper No. 8, Karin et al. disclose a method of treating a cell proliferative disorder associated with JNK comprising administering to a subject with the disorder a therapeutically effective amount of reagent which modulates JNK (see column 12, lines 10-14 and claims 1-2). As further argued, Karin et al. disclose the reagent is an antisense polynucleotide (see column 7, lines 21-39 and claim 2). As further argued, Karin et al. further disclose antisense oligonucleotides of about 15 nucleotides are preferred, since they are easily synthesized (see column 8, lines 58-60). As further argued, Karin et al. further disclose, for example, the method may be useful in treating malignancies of various organ systems, such as lung, prostate cancer, cell proliferative diseases and other types of acute inflammation (see column 12, lines 28-34).

Further, as Applicants have noted, Karin et al. teach polynucleotides which encode the JNK polypeptide, including JNK2. Using the JNK2 polynucleotide disclosure of Karin et al., one of ordinary skill in the art would have expected success in making antisense oligonucleotides since at the time of filing of the instant application, making antisense oligonucleotides was well known in the art. And further since Karin et al. disclose that antisense oligonucleotides of about 15 nucleotides are preferred, since they are easily synthesized, one of ordinary skill in the art would have anticipated the production of the JNK2 antisense oligonucleotides of the current

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invention. It is noted that the instant specification does not disclose more than the disclosure of Karin et al. Thus, Karin et al anticipate the current invention.

The 35 U.S.C. 103(a) rejection over claims 14, 21, 22, and 28-33 as being unpatentable over Semiya et al., Barachinni et al. [U.S. Patent No. 5801154] Shibahara et al., (Nucleic Acids Research, 1989 Vol. 17:239-52), Kallunki et al. (Genes and Development, 1994 p 2996-3007) and Karin et al. [U.S. Patent No. 5837244] is withdrawn in view of Applicant's Declaration under rule 1.131 and Applicant's arguments filed January 6, 2003 in Paper No. 10.

The 35 U.S.C. 103(a) rejection over claims 14, 21, 22, and 28-33 as being unpatentable over Derijard et al. (Cell, 1994 Vol. 76:1025-1037), in view of Karin et al. [WO 95/03324] and Milligan et al. (Journal of Medicinal Chemistry, 1993. 36:1923-1937) is moot in view of Applicant's Amendment to cancel claims 1, 16, and 18, filed January 6, 2003 in Paper No. 10.

The 35 U.S.C. 103(a) rejection over claims 14, 21, 22, and 28-33 as being unpatentable over Derijard et al., Karin et al. and Milligan et al., as taken together and applied to claims 1, 14, 16 and 18 above, and further in view of Shibahara et al. and Kallunki et al. is moot in view of Applicant's Amendment to cancel claims 2-9 and 19, filed January 6, 2003 in Paper No. 10.

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Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 14, 21, 22, and 28-34 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

The instant claims read on a method of modulating the expression of human JNK2 protein in cells or tissues comprising contacting said cells or tissues with an oligonucleotide from 8 to 30 nucleotides in length targeted to a nucleic acid molecule encoding JNK2, wherein said oligonucleotide specifically hybridizes with and inhibits the expression of JNK2.

The claimed invention encompasses any oligonucleotide that specifically hybridize to any form of the JNK2 gene, which includes sequences from any species, mutated sequences, polymorphic and allelic variants, splice variants, sequences that have an unspecified degree of identity (similarity, homology), and so forth. The specification as filed provides only a description of human JNK2 genes (see Tables 8-13).

The specification provides only antisense compounds complementary to target sites, or "targeting" (see specification page 11, lines 7-35 and page 12, lines 1-14) of the human JNK2 mRNA molecule, wherein such antisense compounds are effective to inhibit expression of the target sequence. However, the specification as filed, does not provide sufficient description that would allow one of skill in the art to use Tables 8-13 to predict the structures of antisense

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oligonucleotides complementary to target sites or "targeting" of JNK2 isolated from other sources, including all polymorphic, allelic and splice variants of this mRNA.

The specification fails to describe the complete structure of a representative number of species of the claimed genus. See the Guidelines for Examination of Patent Applications Under the 35 USC 112 ¶ 1, "Written Description" Requirement (Vol. 66, No. 4, pages 1099-1111). These guidelines state that: "To satisfy the written description requirement, a patent specification must describe the claimed invention in sufficient detail that one skilled in the art can reasonably conclude that the inventor had possession of the claimed invention. An applicant shows possession of the claimed invention by describing the claimed invention with all of its limitations using such descriptive means as words, structures, figures, diagrams, and formulas that fully set forth the claimed invention. Possession may be shown in a variety of ways including description of an actual reduction to practice, or by showing that the invention was "ready for patenting" such as by the disclosure of drawings or structural chemical formulas that show that the invention was complete, or by describing distinguishing identifying characteristics sufficient to show that applicant was in possession of the claimed invention." In the instant case, the specification does not describe or identify characteristics that can be used to distinguish species of the claimed genus.

Additionally, "[T]he skilled artisan cannot envision the detailed chemical structure of the encompassed polynucleotides and/or proteins, regardless of the complexity or simplicity of the method of isolation. Adequate written description requires more than a mere statement that it is part of the invention and reference to a potential method for isolating it. The nucleic acid itself is required. See Fiers v. Revel, 25 USPQ2d 1601, 1606 (CAFC 1993) and Amgen Inc. V. Chugai

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Pharmaceutical Co. Ltd., 18 USPQ2d 1016. In Fiddes v. Baird, 30 USPQ2d 1481, 1483, claims directed to mammalian FGF's were found unpatentable due to lack of written description for the broad class. The specification provided only the bovine sequence."

Applicant's specification does not provide a sufficient number of representative species of complementary nucleic acid molecules that target JNK2, which would allow one of skill in the art to predict the structures of all members of the claimed genus of compounds. One of skill in the art would reasonably conclude that the disclosure fails to provide a representative number of species to describe the genus. Therefore, the specification does not describe the claimed compounds in such full and concise terms so as to indicate that the applicant had possession of these compounds at the time of filing of this application. Applicant is reminded that Vas-Cath makes clear that the written description provision of 35 USC 112 is severable from its enablement provision. (See page 1115.).

Claims 14, 21, 22, and 28-34 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method of inhibiting the growth of a breast tumor in an animal comprising the direct administration or intravenous injection of SEQ ID NO: 31 that targets and inhibits the expression of human JNK2 and inhibits the growth of said breast tumor, does not reasonably provide enablement for a method of inhibiting the growth of any tumor in an animal using any oligonucleotide 8 to 30 nucleotides in length that targets and inhibits the expression of human JNK2 by any route of administration. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention commensurate in scope with these claims.

Claim 14 is drawn to a method of modulating the expression of JNK2 in cells or tissues using an oligonucleotide 8 to 30 nucleotides in length that targets and inhibits the expression of human JNK2. Claims 21 and 22 are drawn to a method of inhibiting the growth of a tumor in an animal comprising the administration of an oligonucleotide 8 to 30 nucleotides in length that targets and inhibits the expression of human JNK2. Claims 28-34 are drawn to a method of treating an animal having a disease or condition associated with a human JNK2 protein using an oligonucleotide 8 to 30 nucleotides in length that targets and inhibits the expression of human JNK2.

The instant invention specification provides methodologies for antisense inhibition of human JNK2 in cell culture (see Example 4 and Tables 8-13) and treatment of human breast tumors in mice intravenously injected with an oligonucleotide of SEQ ID NO: 31 targeted to JNK2 protein (see Example 7 and Table 18).

Davis, RJ (Cell, 2000 Vol. 103:239-252) asserts that although the function for JNK is implicated in cancer, the mechanism of JNK action is unclear (see page 247, last paragraph). Davis, RJ further asserts that the basic molecular mechanism of JNK is unknown and future studies are needed to elucidate the function of JNK and the role of JNK in cancer (see page 248, concluding remarks).

The assertions of Davis, RJ indicate that further research is required in the art to understand the function of JNK2 in normal and cancer cells.

Furthermore, the unpredictability of the art of antisense therapy in general adds to the lack of enablement for the current invention. For example, Branch (TIBS, February 1998 Vol. 23, pages 45-50) addresses the unpredictability and the problems faced in the antisense art with

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the following statements: "Antisense molecules and ribozymes capture the imagination with their promise of rational drug design and exquisite specificity. However, they are far more difficult to produce than was originally anticipated, and their ability to eliminate the function of a single gene has never been proven."; "To minimize unwanted non-antisense effects, investigators are searching for antisense compounds and ribozymes whose target sites are particularly vulnerable to attack. This is a challenging quest."; "However, their unpredictability confounds research application of nucleic acid reagents."; "Non-antisense effects are not the only impediments to rational antisense drug design. The internal structures of target RNAs and their associations with cellular proteins create physical barriers, which render most potential binding sites inaccessible to antisense molecules."; "Years of investigation can be required to figure out what an 'antisense' molecule is actually doing..."; "Because knowledge of their underlying mechanism is typically lacking, non-antisense effects muddy the waters."; "Because biologically active compounds generally have a variety of effects, dose-response curves are always needed to establish a compound's primary pharmacological identity. Antisense compounds are no exception. As is true of all pharmaceuticals, the value of a potential antisense drug can only be judged after its intended clinical use is known, and quantitative information about its dose-response curve of conventional drugs, which typically span two to three orders of magnitude, those of antisense drugs, extend only across a narrow concentration range."; "Because it is very difficult to predict what portions of an RNA molecule will be accessible *in vivo*, effective antisense molecules must be determined empirically by screening large number of candidates for their ability to act inside cells."; "Binding is the rare exception rather than the rule, and antisense molecules are excluded from most complementary sites. Since accessibility cannot be

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predicted, rational design of antisense molecules is not possible.”; and, “The relationship between accessibility to oligonucleotide (ODN) binding and vulnerability to ODN-mediated antisense inhibition *in vivo* is beginning to be explored...It is not yet clear whether *in vitro* screening techniques...will identify ODN’s that are effective *in vivo*.”

Jen et al. (Stem Cells, 2000, Vol. 18:307-319) discuss antisense-based therapy and the challenges that remain before the use of antisense becomes routine in a therapeutic setting. Jen et al. discuss the advances made in the art but also indicate that more progress needs to be made in the art. In the conclusion of their review, Jen et al. assert, “Given the state of the art, it is perhaps not surprising that effective and efficient clinical translation of the antisense strategy has remained elusive.” It is also stated, “The key challenges to this field have been outlined above. It is clear that they will have to be solved if this approach to specific antitumor therapy is to become a useful treatment approach. A large number of diverse and talented groups are working on this problem, and we can all hope that their efforts will help lead to establishment of this promising form of therapy.” It is clear from Jen et al. that the state of the art of antisense is unpredictable and those highly skilled in the art are working towards making the art of antisense therapy more predictable but have many obstacles to overcome.

Dias et al. (European Journal of Pharmaceutics and Biopharmaceutics, 2002 Vol. 54:263-269) addresses the limitations of antisense-based therapy. Dias et al. state, “Even though the antisense strategy is widely employed currently, it has certain defined limitations. Although it is relatively easy to synthesize phosphodiester oligonucleotides, these cannot [*emphasis added*] be used as drugs due to their propensity to be easily degraded by cellular nucleases” (see page 263, first column). Dias et al. further discuss that different methods, such as electroporation,

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microinjection or the binding to particular peptides with membrane translocation properties have been developed to overcome internalization problems, however these methods are easily applied in cultured cells, but may or may not be useful in *in vivo* systems (see page 263, second column).

In view of the unpredictability in the art, the specification as filed does not provide adequate guidance or examples that would show by correlation how one skilled in the art would practice the claimed invention over the scope claimed without having to engage in trial and error or undue experimentation. The specification as filed contemplates the therapeutic use of JNK2 antisense in a broad range of divergent/unrelated diseases (e.g. inflammation, fibrosis, fibrotic scarring, peritoneal adhesions, lung fibrosis, conjunctival scarring, hyperproliferative disease, and cancer) using any JNK2 antisense and divergent routes of administration (e.g. intravitreal, intraventricular, intraluminal, intrathecal or intravesical). However, the instant specification does not show any specific link between JNK2 and any specific disease or condition such that treatment with JNK2 antisense would be an apparent treatment option. It is unclear how the specific and direct treatment of human breast tumors in mice with an oligonucleotide of SEQ ID NO: 31 targeted to JNK2 protein data is correlated with/or representative of treatment to any disease or condition with any JNK2 antisense. It is also unclear how any JNK2 antisense will treat any one disease or condition where no specific guidance (i.e. specific mode of treatment, delivery route, tissue specificity, etc.) is provided. It is also unclear how the direct administration or intravenous injection of SEQ ID NO: 31 that targets and inhibits the expression of human JNK2 in mice is representative of other routes of administration (e.g. intravitreal, intraventricular, intraluminal, intrathecal or intravesical) where no specific guidance is provided.

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The specification does not provide particular guidance or particular direction for the treatment of a disease or condition associated with JNK2 in an animal. The specification does not provide guidance for the delivery of antisense compounds into the target organ and target cells in an animal in quantity sufficient to inhibit JNK2 expression. While the specification provides guidance to addressing antisense compound administration to cells in culture and treatment of human breast tumors in mice intravenously injected with an oligonucleotide of SEQ ID NO: 31 targeted to JNK2 protein, the specification provides no particular nexus between the inhibition of JNK2 *in vivo* for the treatment of a disease or condition associated with JNK2 in an animal by intravitreal, intraventricular, intraluminal, intrathecal or intravesical routes of administration, as contemplated by the specification. The specification provides no particular guidance of direction for addressing the problems of targeting, permanence and quantity of expression of the gene in question, immunogenicity, etc, for nucleic acid/antisense targeting JNK2 in an animal. The specification provides no particular guidance or direction for the treatment of an animal having a disease or condition associated with JNK2 using the JNK2 antisense oligonucleotides of the claimed invention. Therefore, in view of the breadth of the claims and the lack of guidance provided by the specification as well as the unpredictability of the art, one of ordinary skill in the art at the time of the invention would have required an undue amount of experimentation to make and use the claimed invention commensurate with the full scope of the claims. Due to the lack of specific guidance in the specification as filed and the lack of correlation between a method of treating human breast tumors in mice intravenously injected with an oligonucleotide of SEQ ID NO: 31 targeted to human JNK2 protein and a method of inhibiting the growth of any tumor in an animal comprising the divergent route of administration

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(e.g. intravitreal, intraventricular, intraluminal, intrathecal or intravesical) of any oligonucleotide 8 to 30 nucleotides in length that targets and inhibits the expression of human JNK2, one of skill in the art would require specific guidance to practice the current invention. The current specification does not provide such guidance to a method of inhibiting the growth of any tumor in an animal comprising the administration of any oligonucleotide 8 to 30 nucleotides in length that targets and inhibits the expression of human JNK2 and one of skill in the art would be required to perform trial and error or undue experimentation. The quantity of experimentation required to practice the invention over the scope claimed would include the de novo determination of how to engineer and deliver any antisense targeting JNK2 such that any disease or condition (e.g. inflammation, fibrosis, fibrotic scarring, peritoneal adhesions, lung fibrosis, conjunctival scarring, hyperproliferative disease, and cancer) associated thereto would be treated to any degree, particularly, in view of the obstacles needed to overcome to use antisense therapies as exemplified in the references discussed above. It is noted that the unpredictability of a particular area may alone provide reasonable doubt as to the accuracy of the broad statement made in support of enablement of claims. See *Ex parte Singh*, 17 USPQ2d 1714 (BPAI 1991). Accordingly, limiting the scope of the claimed invention to a method of inhibiting the growth of a breast tumor in an animal comprising the direct administration or intravenous injection of SEQ ID NO: 31 that targets and inhibits the expression of human JNK2 and inhibits the growth of said breast tumor is proper.


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Any inquiry concerning this communication or earlier communications from the examiner should be directed to Terra C. Gibbs whose telephone number is (703) 306-3221. The examiner can normally be reached on M-F 8:30-5:00.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, John L. LeGuyader can be reached on (703) 308-0447. The fax phone numbers for the organization where this application or proceeding is assigned are (703) 746-8693 for regular communications and (703) 872-9307 for After Final communications.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to the receptionist whose telephone number is (703) 308-0196.

tcg
May 15, 2003


RAM SHUKLA
PRIMARY EXAMINER